

der, causing inability to use the joint. The deltoid, supra-spinatus, infra-spinatus, pectoralis major (slightly), triceps, brachialis anticus, and supinator longus were affected, reacting only slightly to faradism, the deltoid not at all. The faradic current was slowly bringing about a cure.

The second case, a man of thirty-seven, was pitched from a cab, falling on the left shoulder and side of the head, stretching and straining the neck. He gradually lost the use of the arm. About the shoulder there was some anaesthesia at first. The biceps, coraco-brachialis, brachialis anticus, deltoid, and supra-spinatus were completely paralyzed. To strong faradism there was no reaction, the muscles giving the reaction of degeneration. A stretching of the brachial plexus is the cause of nearly all such paralyses, this stretching corresponding to a lesion of the fifth and sixth cervical nerves before joining the plexus. In the monkey, paralysis of the biceps, supinator longus, and deltoid follows the division of these nerves.

A woman, falling asleep with the neck resting against the edge of the table, found herself in this same unhappy plight on awaking. The same thing might happen in infantile paralysis; without anaesthesia, however, the lesion being situated in the anterior horns.

In answer to questions, Dr. Beevor, who exhibited the last case, said there had been no pupillary phenomena, which could only occur if the second dorsal nerve was also affected. He believed that the supinator longus was not a pronator or a supinator, but a pure flexor, and that the action of a muscle produced by faradism was not the same as that produced by the will.

MALARIAL NEURITIS AND NEURO-RETINITIS.

The "British Medical Journal" (March 9, 1890) has a paper on this subject, by N. C. Macnamara. In places where malaria prevails, says the author, hemicrania, sciatica, and loss of sight, in connection with intermittent fever, are not unfrequently met with. A case cited, a former tea-planter in Assam, had frequent attacks of intermittent fever; and several times, accompanying such attacks, impairment of vision that prevented reading and writing for ten days or a fortnight. An attack of ague in England produced in a few hours marked disturbances of vision. With the right eye he could only decipher the letters of Snellen 2.25; and with the left fingers could only be dimly counted. The pupils acted imperfectly to light, and were somewhat dilated. There was no pain, photophobia, or

conjunctival congestion. The optic discs were completely obscured by effusion, which extended into the retina. The retinal veins were tortuous and congested. There was no albumen, no sugar, in the urine. Under quinine, then arsenic and strychnine and a change of air, the discs gradually cleared up. Two months elapsed, however, before the patient could read Snellen 1.25. In six months the optic discs were white; Snellen 0.5 could be read; vision in the right eye was $\frac{1}{2}$, in the left $\frac{1}{2}$; and there was no return of fever.

In another instance, where there was complete blindness—but no suspicion of syphilis, no albumen, no rheumatism—full doses of strychnine and a bracing climate restored sight in about a year. A boy of ten recovered under similar treatment.

Another case added to almost total blindness the annoyances attendant upon paralysis of the left ulnar nerve, together with great difficulty in swallowing. Within three months, under the anti-malarial treatment referred to above, complete power over the nerves and muscles whose functions had been lost was restored. In none of the cases cited was anaemia present, though enlargement of the spleen existed in every one. Kidney trouble, syphilis, and rheumatism were excluded. Impaired vision remained for a long time the only malarial symptom. Except for it, the patients were practically well, unless exposed to sudden cold or damp. Checking the fever prevented the probable atrophy of the disc.

HEREDITY AS A FACTOR IN ALCOHOLISM.

In Paul Sollier's treatise on this subject ("Prix Aubanel," 1889) the following generalities are given as rational conclusions derived from the careful and discriminating study of some 350 families who have numbered one or more idiotic, epileptic, or mentally unsound representatives in the wards of the Bicêtre

I. There exists a form of passion for drink that finds its true place of classification somewhere between dipsomania, hereditary insanity, and acquired alcoholism. This is hereditary alcoholism, more frequent in occurrence than dipsomania and having much in common with acquired alcoholism.

II. Hereditary alcoholism may be identical with its source, or different in its manifestations, the proportion of the first to the second being as three to four.

III. Hereditary alcoholism belongs to neuropathic humanity, particularly to its psychopathic division.